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Original Article

Sleep timing, chronotype, mood, and behavior at an Arctic latitude (69°N)

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ABSTRACT

Objective: Daylight is an important zeitgeber for entraining the circadian rhythm to a 24 h clock cycle, especially within the Polar circle, which has long Polar nights several months each year. Phase delays in sleep timing may occur, but the mean shift is normally small. However, the individual variation in phase shifts is large, implicating moderating factors. Here we examined the role of several self-regulatory variables (mood and fatigue, behavioral habits, and psychological self-regulation) as moderators of seasonality in sleep timing and chronotype.

Methods: A sample of 162 young adults (76% females; mean age: females 23.4 years, males 24.3 years) participated in a prospective study across three seasons (September, December, March) in Tromsø/Norway at 69°39′N. Sleep diary and sleep/health-related questionnaire data were collected at each time-point.

Results: Sleep timing and chronotype were delayed during the dark period (December) compared with brighter photoperiods (September and March). Comparable effects were observed for insomnia, fatigue, mood (depression and anxiety), subjective health complaints, physical activity, and school-related stress. Most importantly, depression and fatigue moderated the degree of seasonal shifting in sleep timing, whereas the other self-regulation indicators did not (ie eating habits, physical activity, and psychological self-regulation).

Conclusion: Seasonality in sleep timing and chronotype was confirmed, and it seems that depressive symptoms during the dark period exacerbate phase-shifting problems for people living in sub-Arctic regions.

1. Introduction

People living in sub-Arctic regions may experience more seasonal variations in sleep patterns and problems [1] than people living closer to the Equator [2]. Chronotype also seems more delayed in people living at higher latitudes [3,4], but seasonal variations in chronotype are less well studied. A single cross-sectional study is available; however, it indicates a small magnitude for chronotype changes [5]. The hypothesized explanation is that one of the two processes regulating sleep – the endogenous sleep circadian factor [6] – becomes more easily desynchronized with the 24 h clock during the winter months when sunlight is absent. The other factor – the internal homeostat – exponentially increases sleep pressure the longer a person is awake, but is not affected by seasons.

The circadian factor is a biologically governed process and several clock genes responsible for the rhythmicity have been identified [7]. Since the circadian clock follows a mean 24.18 h rhythm [8], an ever-increasing mismatch between the environmental and the circadian rhythm is in operation. The circadian clock hence needs to be reset or advanced each day to stay synchronized with the daily spin of the Earth. A morning dose of bright daylight is the most potent zeitgeber (time-giver) for entraining and preventing the circadian clock from free-running [9,10]. When light relayed from the retina hits the suprachiasmatic nuclei in the hypothalamus, the production of melatonin in the pineal gland is suppressed [11]. During the evening, melatonin secretion again increases and induces drowsiness [12], accompanied by reduced alertness, reduction in body temperature, and decreased cardiovascular output [13].

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Since melatonin is important for sleep–wake cycles [14] and is influenced by light, seasonal differences in daylight exposure should influence circadian rhythms and sleep timing. Studies conducted under extreme conditions at base camps in Antarctica confirm that desynchrony is common among inhabitants during winter [15,16]. As the sub-Arctic people are not exposed to bright sunlight during winter, a delay in chronotype is expected [3,10]. However, not all individuals seem to develop large phase shifts, suggesting that additional processes must be involved. The present study examined whether several behavioral or psychological self-regulatory factors played a moderating role of seasonal shifts in sleep timing.

The city of Tromsø (69°39′N) represents a natural habitat for studying regularity in sleep timing and chronotype, as the sun is absent between mid-November and mid-January. Tromsø has a population of about 70,000 and is the largest city in the world at this latitude. Tromsø is comparable to other western cities with regard to modern facilities and living standards. It has a university hospital and a university campus housing about 10,000 students. Mean temperatures range between 11 °C and −4 °C due to the Gulf Stream. Although the sun is absent, it does not become pitch black even at winter solstice. The sky is still deep blue at midday, but the intensity is dim and of short duration (∼3 h).

The typical complaint during the winter months is increased sleeplessness and fatigue. In the population-based cross-sectional 'Tromsø study' (N = 12,984), increased insomnia problems [1] and phase shifts [17] have been reported more often during winter than during summer. 'Winter insomnia' also seems more prevalent among women [18]. These sleep problems may be alleviated by bright light therapy [19,20], or short wavelength (460–480 nm) blue light [21]. In the Tromsø study using a retrospective recall of bed times and wake times, chronotype was less affected by season, delaying by ∼10 min [5]. In a prospective study using sleep diary recordings across two seasons (N = 200 + 150, 7 days, ∼2450 recordings) [2], the phase delay from summer to winter was ∼20 min. The prospective design thus seems to detect larger phase shifts, and in the present study we extended the repeated measures design by adding a third season to examine this further.

The individual variation in delay is, however, very large (±90 min, 68% confidence interval) [2]. Individuals may accumulate a considerable amount of sleep debt during work days and develop 'social jetlag' (ie mismatch between circadian and social rhythms). Thus, it is necessary to examine to what extent various hypothesized self-regulatory behaviors may account for the large variations in sleep timing when bright morning light is absent.

1.1. Self-regulation and sleep

In the absence of sunlight during winter, self-regulation skills may help in offsetting delays in sleep timing and other sleep problems. Self-regulation is considered a vital aspect of many mental health problems or disturbances [22]. Direct measures of self-regulation are, however, not readily available due to its extensive scope and multifaceted nature. Self-regulation closely resembles concepts such as willfulness, self-control, motivation, inhibition, executive control, and impulse control, to mention a few. An underlying common factor is the ability to plan, guide, and monitor one's behavior flexibly, to sustain task performance, and to maintain a certain degree of regularity [22].

Seasonal shifts in sleep are accompanied by moderate to strong seasonal changes in fatigue, of which physical fatigue seems more influenced than mental fatigue [2]. Fatigue is also negatively correlated with physical activity and regularity [23], and may therefore act as a moderator of sleep timing. Negative mood and depression symptoms, especially, show a similar albeit weaker pattern [1,2,15,24,25]. Depressed mood should not be confused with 'winter depression' or, more specifically, seasonal affective disorder [26,27]. Nevertheless, symptoms of depression and anxiety represent important indications of dysregulation in mood and cognitions [28], and are related to poorer sleep quality [29].

Self-regulation and motivation may also regulate sleep behaviors. The ability to plan ahead and control one's impulses is important for good mental health and general adaptability [30–32]. According to the regulatory focus theory of Higgins [33], behavior may be motivated for two reasons: to prevent negative health outcomes and/or to promote desired goals (like better sleep). Regulatory focus and sleep have not been previously examined, but, based on how regulatory focus generally predicts motivation [34] and is related to consumption of healthy food [35], it may be associated with better self-regulation of sleep patterns as well.

Eating habits and frequency in physical activity also indicate self-regulation, as both represent behavioral indications of regularity. Unhealthy or unstable eating habits have been connected with a delay in chronotype [36] and with poorer sleep, as instability in meals is associated with more frequent headaches [37], which impact negatively on sleep. Phase shifts influenced by daytime physical activity have little empirical support, but may be expected to influence sleep timing similarly.

1.2. Hypotheses

(1) Based on sleep diary data, sleep timing (sleep onset and wake times) and chronotype are hypothesized to covary with seasons, displaying significant phase delays during the dark period (December) compared to brighter photoperiods (September and March).
(2) Insomnia, depression, fatigue, and anxiety will show a similar seasonal pattern as described in hypothesis 1.
(3) The strength of the correlation between season and sleep timing/chronotype (hypothesis 1) will be modified by indices of self-regulation, ie individuals characterized by better self-regulation will demonstrate less phase delay during the dark period relative to individuals demonstrating poorer self-regulation.

2. Methods

2.1. Participants

The participants (N = 162) consisted of a convenience sample recruited during lectures at the University of Tromsø in Norway in December 2011 (n = 94; 70/24 females/males) and in March 2012 (n = 68; 54/14 females/males). The difference in mean age of females (mean, 23.4; standard deviation, 5.0; range, 19–48) and of males (24.3; 5.4; 19–46) was not significant. Females were over-represented (76%) compared with the number enrolled at the University in 2012 (60%) [38]. The participation (response rate) from wave 1 to wave 3 fell as follows: 162 (100%), 148 (92%), and 138 (85%), respectively. Participants were thus reordered according to seasons: September (T1, N = 138, 77% females), December (T2, N = 148, 76% females) and March (T3, N = 150, 78% females). Among the 162 students, 58 studied medicine and odontology (36%), 46 studied psychology (28%), and the remaining 58 students (36%) followed courses within social sciences, economy, or leadership.

The study was approved by the regional ethical committee for medical and health research (case ID: 2011/743/REK Nord), and conformed to international ethical standards [39].

2.2. Study design

The study used a within-subjects, quasi-experimental design assessing sleep and questionnaire data across three seasons

(December, March and September). To control for sequence effects, students were recruited in two different waves (December and March), and used as a covariate to separate effects related to order. Hence the design had a partial counterbalancing scheme.

2.3. Procedures

Data on sleep were recorded in a sleep diary that the students kept for one week and completed at home. A questionnaire package was also completed at home during the same week, but before completing the sleep diary. Participants received a payment of 250 Norwegian krona (about US $41) after each data collection wave.

2.4. Measurement of demographics

Questions about age, gender, place of birth, length and type of education, marital status, and number of children were included.

2.5. Sleep diary parameters

Sleep data were collected via a seven-day sleep diary. Data were averaged for work days and free days, including time going to bed (BT), lights out time (LT), sleep onset latency (SOL, min), duration of nightly waking after sleep onset (WASO, min), waking-up time (WUT), and out of bed time (OUT). Sleep onset time (SOT) was calculated as LT + SOL, total time in bed (TIB) as OUT – BT, early morning awakening (EMA) as OUT – WUT, total sleep time (TST) as WUT – SOT – WASO, and sleep efficiency (SE,%) as TST/TIB × 100 [40].

Chronotype was estimated according to Roenneberg et al. [41] as the mid-point between sleep onset and wake time for free days (MSF) as collected from the sleep diaries. The mid-time for work days (MSW) was calculated similarly. The differences in sleep duration during work days and free days were used to make an adjusted $MSF_{sc} = MSF - (TST_{free\ days} - TST_{work\ days})/2$ as people tend to accumulate sleep debt during the week. Social jet lag was calculated as MSF – MSW.

2.6. Sleep- and mood-related questionnaires

2.6.1. Bergen Insomnia Scale (BIS)

The BIS was used to measure insomnia symptoms, and is based on the Diagnostic and statistical manual of mental disorders, 4th ed., text revision (DSM-IV-TR) criteria for insomnia. Three items cover nocturnal symptoms, and another three items cover daytime function problems. Each item is scored as number of days the problem was present (range, 0–7), hence indicating the frequency of insomnia symptoms. Total scores range from 0 to 42 [42]. The reliability (Cronbach's α) in each season (September, December and March) was 0.75, 0.67 and 0.78, respectively.

2.6.2. Fatigue Questionnaire (FQ)

The FQ consists of 11 items. Seven items measure physical fatigue, and four measure mental fatigue. Each item is scored from 0 to 3 (total range, 0–33) with higher scores indicating more fatigue. The scales are suitable for general populations and have good psychometric properties [43]. Cronbach's α across the three seasons was 0.87, 0.83 and 0.86 (physical fatigue) and 0.65, 0.52 and 0.61 (mental fatigue), respectively.

2.6.3. Hospital Anxiety and Depression Scale (HADS)

HADS consists of 14 items assessing non-vegetative symptoms of depression (seven items) and anxiety (seven items), respectively [44]. Item scores range from 0 to 3, and total scores range from 0 to 21 for each subscale. It is widely used as a screening instrument, where a cut-off score $\geqslant 8$ indicates a tentative diagnosis of depression [45]. Cronbach's α was 0.66, 0.74 and 0.73 (depression) and 0.73, 0.76 and 0.75 (anxiety), respectively.

2.7. Behavioral regularity measures

The following questions were included from the large general population-based Nord-Trøndelag HUNT study in Norway [46].

Regularity in eating habits was measured by four frequency questions asking how many days per week participants ate breakfast, lunch, dinner, and supper (range, 0–3; 3: each day; 2: 4–6 days a week; 1: 1–3 days a week; 0: seldom or never), as well as one question about number of meals per day (range, 0–4). An exploratory factor analysis (EFA) extracted a single component (eigenvalues >1) across all three seasons ($\lambda = 2.89$, 2.49 and 2.54), hence supporting the use of a single sum score.

Physical activity was rated with three questions asking for frequency in exercise, intensity, and duration of exercise. A comparable EFA extracted a single component across all seasons ($\lambda = 1.70$, 1.73 and 1.74), supporting the use of a single sum score.

2.8. Psychological Self-Regulation Questionnaires

The 63-item Self-Regulation Questionnaire (SRQ) developed by Brown et al. [47] covers seven dimensions of self-regulation. In the present study, a brief 10-item version based on work by Neal and Carey [48] was used to measure two subfactors: impulse control (six items, eg 'give up easy') and goal setting (four items, eg 'trouble making plans'). Cronbach's α was 0.83, 0.75 and 0.82 (impulse control) and 0.68, 0.62 and 0.69 (goal setting) across the three seasons, respectively.

The Regulatory Focus (RF) Scale of Higgins [33] covers motivational processes to a larger extent. The RF measures two qualitatively different motivational origins using 14 items, the motivation to achieve goals or good health (promotion focus, six items), and to avoid failures or damages (prevention focus, eight items). A promotion focus has been connected with better academic achievements and health [49]. Cronbach's α in the present study was 0.84, 0.82 and 0.83 (prevention) and 0.76, 0.70 and 0.65 (promotion) across the three seasons, respectively.

2.9. Covariate questionnaire measures

Use of alcohol was rated by asking how many glasses (comparable to alcohol units) of red wine, beer and liquor had been consumed during the last two weeks. As people who drink beer also drink liquor ($r = 0.37$), but not red wine ($r = -0.18$), consumption of red wine and beer/liquor were separated in two scores.

Psychosomatic health was measured by the Subjective Health Complaints Questionnaire [50]. This asks for complaints during the last 30 days across five domains: musculoskeletal, pseudo-neurological, gastrointestinal, allergic, and flu-like complaints. The total score was used in the present study as it is a valid and reliable measure of complaints [51]. Cronbach's α in the present study was 0.77, 0.83 and 0.81 across the three seasons, respectively.

Subjective stress related to following a university curriculum was assessed by using two subscales from the Adolescent Stress Questionnaire [52], ie school performance stress (five items) and school attendance stress (five items). All items were scored 1–7; higher scores signify more stress. Cronbach's α was 0.82, 0.80 and 0.80 (performance) and 0.83, 0.81 and 0.79 (attendance) across the three seasons, respectively.

2.10. Statistical analyses

IBM SPSS version 21 was used to conduct descriptive and inferential statistics. We used linear-mixed regression models to avoid

excluding cases with incomplete repeated data. The restricted maximum likelihood procedure and type III *F*-tests were used. A compound symmetry specification of the residual matrix was chosen as other specifications (eg auto-regressive) did not improve model fit. Moderation of seasonal shifts was examined by entering an interaction term (season × variable) based on centered values. Interaction effects were plotted according to ±1 SD of the moderator variable. Least significant difference (LSD) post-hoc tests were used to compare differences between two seasons. Standardized effect sizes were calculated as $d = \frac{t}{\sqrt{df}}$ and interpreted as small ($d = 0.2$), moderate ($d = 0.5$) and strong ($d = 0.8$) according to Cohen [53]. Covariates were included in order to adjust the mean scores for potentially sleep-interfering factors such as age, gender, number of children, marital status, education (years), curricula (any vs medical), birthplace (south vs north), number of days outside the county, daily use of caffeine, alcohol, tobacco or medication (no vs yes), and recruitment sequence (starting the study in December vs March). Non-significant covariates were removed backwardly, hence only adjusting the sleep scores for statistically significant covariates.

3. Results

3.1. Descriptive analyses

The correlation coefficients between the measured variables were based on all seasons combined (Table 1).

3.2. Seasonal differences in sleep timing and chronotype

Both unadjusted and adjusted sleep diary data are presented in Table 2. As expected, the timing of sleep correlated with seasons. The recordings for evening sleep time were significantly delayed in December compared with September/March for bed time ($t = 4.29$, $P < 0.001$; $t = 2.35$, $P < 0.05$), SOL ($t = 4.80$, $t = 3.59$, both $P < 0.001$) and SOT ($t = 6.16$, $t = 4.32$, both $P < 0.001$). The seasonal effect approached a moderate size for SOT ($d = 0.40$). WASO increased from September to December ($t = 2.05$, $P < 0.05$), but did not fall back in March. Wake time was significantly later in December compared with September/March ($t = 5.50$, $t = 5.32$, both $P < 0.001$), and sleep efficiency was worse in December relative to September/March ($t = 4.40$, $t = 3.58$, both $P < 0.001$). The chronotype indicator (MSFsc) showed a seasonal effect by a delay from September to December ($t = 3.52$, $P < 0.001$). The advance in March was significant if using a one-tailed test ($t = 1.63$, $P = 0.052$); however, the unadjusted effect was also significant ($t = 2.03$, $P < 0.05$). TST was unrelated to season.

These seasonal differences in sleep timing were generally most pronounced for work days relative to free days. For example, EMA only showed an expected seasonal difference for work days, being highest in December compared with September/March ($t = 3.67$, $t = 3.46$, both $P < 0.001$).

As some of the questionnaire variables (fatigue, depression and SHC) were weakly correlated with sleep diary data (maximum $r = 0.23$), these questionnaire variables were added as covariates to the above models. However, they had no significant influence on the reported seasonal effects.

3.3. Seasonal differences in the questionnaire data

Problems related to insomnia were more pronounced during December than in September or March ($t = 6.45$, $t = 3.13$, both $P < 0.001$) (Table 3). The seasonal effect was more pronounced for daytime ($d = 0.38$) than nocturnal ($d = 0.22$) symptoms. Fatigue showed a similar pattern, being highest in December compared with September/March ($t = 7.66$, $t = 4.08$, both $P < 0.001$). The effect was stronger for physical ($d = 0.47$) than mental ($d = 0.22$) fatigue.

Seasonal variations in mood and subjective health complaints (SHC) were partly present, with an increase in depression ($t = 5.42$, $P < 0.001$), anxiety ($t = 6.30$, $P < 0.001$), and somatic complaints ($t = 6.01$, $P < 0.001$) from September to December. The weak decline in March was only statistically significant for anxiety and SHC if using one-tailed tests. The stress-related variables were higher in December compared with September/March for both school performance stress ($t = 5.32$, $t = 2.91$, $P < 0.001$, $P < 0.01$), and school attendance stress ($t = 5.10$, $t = 3.30$, both $P < 0.001$). There were no discernible seasonal differences for regularity in eating, physical activity, use of alcohol, and the psychological regulatory focus measure.

As the questionnaire variables were highly inter-correlated, they were entered simultaneously in a multivariate regression to

Table 1
Pearson correlation coefficients between all measured variables on all seasons combined ($N = 559$).

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1 SOL																			
2 SOT	0.36																		
3 WASO	0.20	−0.04																	
4 Wake-up	0.21	0.75	0.03																
5 EMA	0.15	0.13	0.19	0.05															
6 TST	−0.19	−0.23	−0.06	0.47	−0.14														
7 SE	−0.63	−0.32	−0.42	−0.12	−0.60	0.32													
8 Chronotype	0.12	0.70	−0.04	0.72	0.10	0.17	−0.11												
9 Insomnia	0.35	0.16	0.28	0.13	0.13	−0.07	−0.34	0.04											
10 Fatigue	0.20	0.13	0.13	0.13	0.08	0.00	−0.16	0.08	0.56										
11 Depression	0.18	0.11	0.10	0.09	0.06	−0.04	−0.15	0.01	0.40	0.55									
12 Anxiety	0.08	0.11	0.03	0.09	0.05	−0.02	−0.13	0.03	0.34	0.38	0.54								
13 SHC	0.13	0.08	0.23	0.10	0.00	0.00	−0.13	0.07	0.45	0.47	0.40	0.48							
14 RF prev	0.04	−0.14	0.09	−0.15	−0.03	−0.06	0.00	0.00	−0.06	−0.20	−0.31	−0.31	−0.18						
15 RF prom	−0.03	−0.03	−0.02	0.01	−0.11	0.05	0.04	−0.06	0.04	−0.09	−0.11	0.04	0.03	0.31					
16 Eating habits	−0.06	−0.14	0.05	−0.16	−0.08	−0.05	0.08	−0.14	−0.09	−0.12	−0.19	−0.10	−0.09	0.15	0.09				
17 Physical activity	0.01	0.08	−0.03	0.10	−0.07	0.05	0.00	0.14	−0.05	−0.18	−0.10	0.03	−0.16	0.22	0.19	0.14			
18 Stress	0.05	0.14	−0.06	0.16	0.05	0.05	−0.05	0.11	0.27	0.36	0.36	0.48	0.37	−0.34	0.02	−0.15	−0.07		
19 Beer/liquor	0.04	0.32	0.00	0.32	−0.04	0.05	−0.02	0.33	0.01	−0.06	−0.14	−0.11	−0.17	−0.04	−0.05	0.08	0.19	−0.12	
20 Red wine	−0.15	−0.03	−0.06	−0.02	−0.04	0.02	0.12	0.08	0.03	−0.02	−0.09	0.03	0.09	0.00	0.01	0.05	0.01	0.02	−0.06

SOL, sleep onset latency; SOT, sleep onset time; WASO, wake after sleep onset; EMA, early morning awakening; TST, total sleep onset; SE, sleep efficiency; MSFsc, chronotype indicator; SHC, subjective health complaints; RF, regulatory focus.
Correlation coefficients $r \geqslant 0.10$ and $r \geqslant 0.13$ were significant at $P < 0.01$ and $P < 0.001$, respectively.

Table 2
Unadjusted and adjusted marginal means for the sleep diary variables.

Variables	Workdays				Weekend				All days			
	Sep	Dec	Mar	Difference (Cohen's *d*)	Sep	Dec	Mar	Difference (Cohen's *d*)	Sep	Dec	Mar	Difference (Cohen's *d*)
Bed time				aB[c] (0.21), Bc[a] (0.14)				aB[b] (0.18)				aB[c] (0.26), Bc[a] (0.14)
Mean	23:38	23:54	23:43		0:51	1:19	1:06		23:58	0:19	0:06	
SD	0:57	1:06	1:00		1:21	1:36	1:45		0:52	1:04	1:03	
Adj. mean	23:37	23:53	23:43		0:48	1:16	1:04		23:58	0:18	0:08	
SOL (min)				aB[c] (0.29), Bc[c] (0.25)				aB[a] (0.14)				aB[c] (0.32), Bc[c] (0.24)
Mean	18.4	27.5	19.9		13.3	18.2	14.6		16.9	24.5	18.5	
SD	15.5	27.4	14.7		16.6	27.6	17.4		13.8	23.5	12.5	
Adj. mean	18.5	27.6	20.1		13.4	18.5	14.5		17.5	25.4	19.6	
SOT (min)				aB[c] (0.36), Bc[c] (0.31)				aB[c] (0.23)				aB[c] (0.40), Bc[c] (0.28)
Mean	0:16	0:47	0:21		1:22	1:57	1:39		0:34	1:07	0:43	
SD	0:59	1:14	0:59		1:20	1:33	1:38		0:56	1:09	1:02	
Adj. mean	0:21	0:48	0:25		1:20	1:54	1.38		0:43	1:12	0:51	
WASO (min)				aB[a] (0.12)								aB[a] (0.11)
Mean	4.0	5.6	6.3		3.9	4.6	4.3		4.0	5.3	5.7	
SD	7.2	8.8	8.5		8.7	8.3	8.2		6.4	7.7	7.4	
Adj. mean	3.9	5.5	6.1		3.7	4.4	4.1		3.8	5.2	5.6	
Wake time				aB[c] (0.29), Bc[c] (0.32)				aB[c] (0.21), Bc[a] (0.13)				aB[c] (0.36), Bc[c] (0.34)
Mean	7:50	8:23	7:52		9:27	10:04	9:41		8:17	8:52	8:23	
SD	1:09	1:20	1:04		1:34	1:51	1:37		1:04	1:18	1:04	
Adj. mean	7:51	8:18	7:48		9:28	10:01	9:40		8:20	8:49	8:21	
EMA (min)				aB[c] (0.22), Bc[c] (0.21)								
Mean	13.4	18.7	13.8		27.1	21.4	24.7		17.2	19.4	17.3	
SD	13.9	19.4	14.3		36.7	24.8	22.4		16.4	18.5	13.9	
Adj. mean	13.9	19.2	14.2		27.1	21.4	24.7		17.7	19.9	17.2	
TST	7.48	7.51	7.41		8	8.02	7.95		7.63	7.65	7.56	
Mean	0.89	0.96	0.88		1.27	1.37	1.41		0.75	0.89	0.79	
SD	7.50	7.50	7.39		7.91	7.95	7.86		7.34	7.34	7.28	
Adj. mean												
SE (%)				aB[c] (0.31), Bc[c] (0.25)								aB[c] (0.28), Bc[c] (0.23)
Mean	88.9	85.7	88.3		87.9	88.7	88.7		88.7	86.5	88.3	
SD	5.9	7.5	6.3		12.9	9.6	7.4		6.7	6.8	5.6	
Adj. mean	88.4	85.4	87.8		87.8	88.7	88.8		88.5	86	88	
MSFsc												aB[c] (0.21)
Mean									5:09	5:41	5:24	
SD									1:28	1:37	1:41	
Adj. mean									5:05	5:37	5:22	
SJL (h)												
Mean									1.38	1.41	1.56	
SD									1.22	1.42	1.16	
Adj. mean									1.42	1.46	1.63	

SD, standard deviation; Adj., adjusted; SOL, sleep onset latency; SOT, sleep onset time; WASO, awake after sleep onset; EMA, early morning awakening; TST, total sleep time; SE, sleep efficiency; MSFsc, chronotype; SJL, social jet lag.
[a] $P < 0.05$.
[b] $P < 0.01$.
[c] $P < 0.001$.

determine their unique contribution to seasonality (Table 4). Physical fatigue was the only variable showing seasonal shifts independently of the other variables. As the fatigue variable most frequently rendered all the other variables non-significant, it appears most sensitive to seasonal change.

3.4. Moderator analyses

In the final analyses, we examined whether the seasonal shifts in the sleep parameters for all days combined were modified by the self-regulation variables (Table 5). A statistically significant interaction was detected for depression, fatigue, and anxiety as modifiers. Depression received most support as it significantly modified bed time, SOT, wake time and EMA, whereas fatigue and anxiety modified SOT and wake time only. Seasonality in EMA was modified by depression only, showing a seasonal change (September, 16 min; December, 21 min; March, 15 min) in the +1 SD depression group but minimal changes (September, 18 min; December, 17 min; March, 19 min) in the −1 SD group. Since SOT and wake time were the most relevant variables for sleep timing, all three moderators were regarded as significant modifiers of sleep timing (Fig. 1). If a more stringent α-level had been chosen ($P < 0.01$), only depression would have been significant. Sleep onset time was most strongly modified by depression, but less so by fatigue and anxiety. Seasonality in sleep onset was minimal across seasons (September, 00:36; December, 00:51; March, 00:42) for those with low/no depressive symptoms (−1 SD), but more pronounced in the +1 SD group (September, 00:32; December, 01:17; March, 00:44), being 45 min later in December compared with September. The results for wake times were comparable, but the differences were less pronounced. The three-way interaction (season × depression × fatigue) was not significant.

Table 3
Unadjusted marginal mean (SD) values for the questionnaire data.

Variables	September	December	March	Difference (Cohen's *d*)
Insomnia	9.33 (5.45)	12.42 (5.71)	10.97 (6.38)	aB[c] (0.39), Bc[b] (0.19)
Nocturnal	2.90 (2.54)	3.75 (2.92)	3.37 (3.16)	aB[c] (0.22)
Daytime	6.41 (3.96)	8.67 (4.26)	7.59 (4.37)	aB[c] (0.38), Bc[b] (0.19)
Fatigue	12.22 (4.24)	15.63 (4.27)	13.86 (4.77)	aB[c] (0.45), Bc[b] (0.24)
Physical	7.87 (3.40)	10.75 (3.38)	9.08 (3.61)	aB[c] (0.47), Bc[c] (0.28)
Mental	4.34 (1.37)	4.88 (1.43)	4.78 (1.62)	aB[c] (0.22)
Depression	1.90 (2.04)	3.09 (2.85)	2.97 (2.77)	aB[c] (0.32)
Anxiety	4.75 (2.99)	6.30 (3.53)	5.88 (3.41)	aB[c] (0.37)
SHC	8.73 (6.36)	11.49 (7.95)	10.69 (7.43)	aB[c] (0.37)
Eating habits	2.43 (0.55)	2.38 (0.53)	2.40 (0.53)	
Physical activity	1.05 (0.34)	1.10 (0.32)	1.13 (0.30)	aB[a] (0.12)
RF prevention	3.96 (0.69)	3.90 (0.63)	3.93 (0.70)	
RF promotion	3.81 (0.60)	3.74 (0.60)	3.79 (0.55)	
Glasses red wine	2.75 (3.05)	2.53 (2.76)	2.90 (3.27)	
Glasses beer/liquor	4.05 (6.25)	3.51 (5.80)	3.79 (6.36)	
Stress performance	3.75 (1.30)	4.25 (1.29)	3.98 (1.31)	aB[c] (0.32), Bc[b] (0.17)
Stress attendance	3.00 (1.35)	3.47 (1.35)	3.17 (1.29)	aB[c] (0.30), Bc[c] (0.20)

SD, standard deviation; SHC, subjective health complaints; RF, regulatory focus.
[a] $P < 0.05$.
[b] $P < 0.01$.
[c] $P < 0.001$.

Table 4
Multivariate adjusted marginal mean (SE) values for the questionnaire data.

Variables [covariates]	September	December	March	Difference
Insomnia [+4, +6, +9]	10.67 (0.42)	11.42 (0.40)	11.09 (0.40)	
Nocturnal [+7, +9]	3.20 (0.23)	3.57 (0.23)	3.33 (0.22)	
Daytime [+6, +9, +11]	7.44 (0.29)	7.87 (0.29)	7.73 (0.28)	
Fatigue [+5, +7, +9, +11]	13.38 (4.24)	14.80 (4.27)	13.70 (4.77)	aB[b] (0.21), Bc[a] (0.18)
Physical [+5, +7, +9]	8.68 (3.40)	10.16 (3.38)	8.93 (3.61)	aB[b] (0.26), Bc[b] (0.24)
Mental [+5, +7, +9, +11]	4.68 (1.37)	4.66 (1.43)	4.76 (1.62)	
Depression [+2, +6, +8]	2.53 (2.04)	2.57 (2.85)	2.93 (2.77)	
Anxiety [−3, +7, +9, +10]	5.38 (2.99)	5.86 (3.53)	5.62 (3.41)	
SHC [−1, +2, +6, +7, +8]	9.46 (6.36)	9.75 (7.95)	9.74 (7.43)	
Stress performance [−1,+8,+11]	3.80 (1.30)	4.01 (1.29)	3.89 (1.31)	
Stress attendance [+3,+6,+8,+10]	3.22 (1.35)	3.19 (1.35)	3.17 (1.29)	

SE, standard error; SHC, subjective health complaints; HADS, Hospital and Anxiety and Depression Scale.
Covariates: 1, gender (women/men); 2, age; 3, No. of children; 4, marital status (married/single); 5, insomnia; 6, fatigue; 7, HADS depression; 8, HADS anxiety; 9, SHC; 10, school stress 1; 11, school stress 2.
Covariates that were significantly negatively (ex: [−1]) or positively (ex: [+6]) related with the outcome variable are indicated in superscript.
[a] $P < 0.01$.
[b] $P < 0.001$.

Table 5
Statistically significant moderator variables of seasonality in the sleep diary data.

Variables	HADS depression			Fatigue			HADS anxiety		
	S	M	S × M	S	M	S × M	S	M	S × M
Bed time	8.20[d]		3.09[b]	5.33[c]			8.77[d]		
SOL	9.89[d]	8.03[c]		7.03[d]	12.46[d]		12.41[d]		
SOT	19.95[d]		4.75[c]	14.32[d]	5.76[b]	2.72[a]	20.64[d]		3.40[b]
WASO									
Wake time	19.72[d]		3.87[b]	13.22[d]		3.39[b]	19.28[d]		3.63[b]
EMA			4.34[b]						
TST									
SE (%)	5.35[c]			3.59[b]	8.36[c]		5.14[c]		
MSF_{sc}	5.83[c]			4.41[c]			5.12[c]		2.69[a]

HADS, Hospital and Anxiety and Depression Scale; S, season; M, moderator (e.g. HADS depression); S × M, season × moderator; SOL, sleep onset latency; SOT, sleep onset time; WASO, awake after sleep onset; EMA, early morning awakening; TST, total sleep time; SE, sleep efficiency; MSF_{sc}, chronotype.
The following variables were additional examined as moderators of sleep timing, but found non-significant: eating habits, physical activity, regulatory focus (RF) prevention and RF promotion.
[a] $P < 0.07$.
[b] $P < 0.05$.
[c] $P < 0.01$.
[d] $P < 0.001$ (*F*-test).

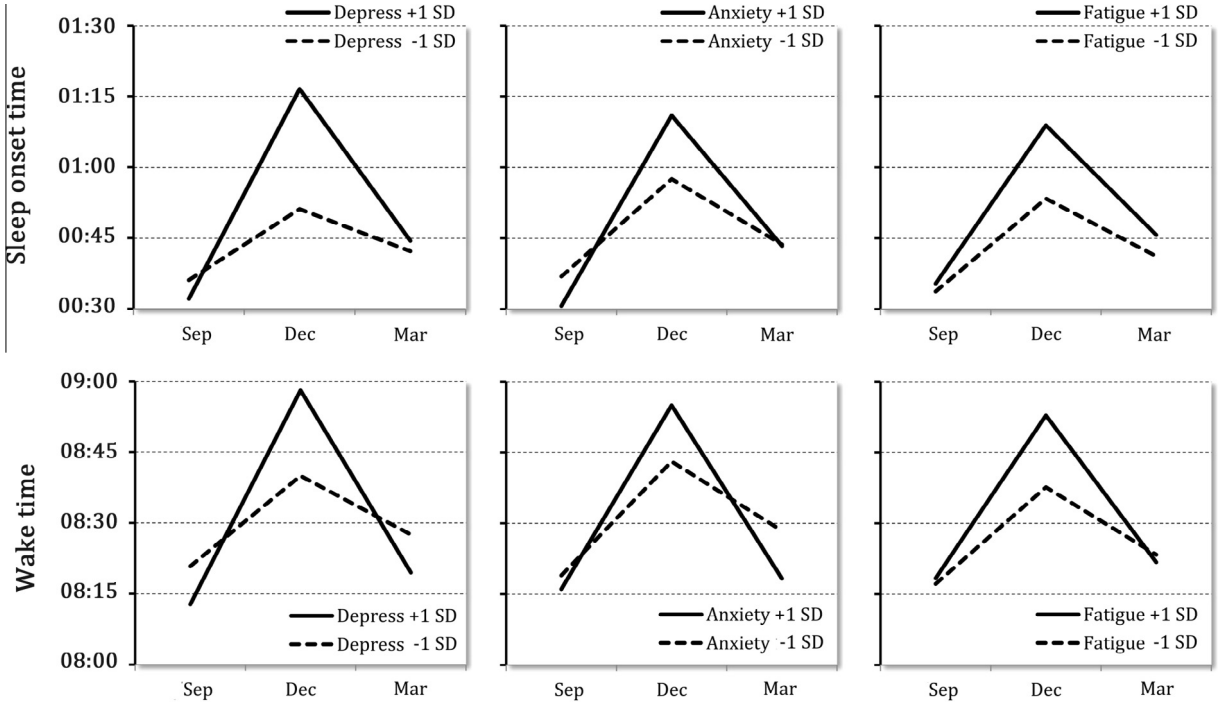

Fig. 1. Graphs for sleep onset time and wake time across three seasons depending on low (−1 SD) or high (+1 SD) scores on the Hospital Anxiety and Depression Scale (HADS)–Depression, HADS–Anxiety, or the Fatigue Scales.

The other interaction tests for daily eating habits, physical activity and the psychological self-regulatory measures (SRQ and RF) showed no conditional influence on the sleep diary data.

The above analyses were repeated for work days and free days (ie weekends) separately, using depression as the only moderator. The interaction terms for WASO and TST remained non-significant for work days and free days. Bed time lost its significance as the interaction terms for both work days and free days were non-significant. SOT, wake time, and EMA were significant only for work days ($P = 0.02$, $P = 0.03$, and $P = 0.01$, respectively), but not for free days ($P = 0.12$, $P = 0.38$, and $P = 0.11$, respectively). The interaction term for SE% was non-significant for all days, but was close to significant for work days ($P = 0.06$). Taken together, depressive symptoms acted as a significant moderator of sleep timing for work days but were irrelevant during free days.

4. Discussion

The present study confirmed several previous findings on seasonality in sleep timing among people living within the Arctic Circle. We observed that individuals went to bed about 21 min later, went to sleep 34 min later, and woke up 35 min later during the dark period (December) compared with brighter photoperiods (September and March). In comparison with our previous study [2], bed time was slightly more delayed in the present study (20 vs 12 min) whereas rise time (wake time + EMA) was equally delayed (32 min in both studies). These delays across seasons coincide with previous findings [54,55]. Despite a delay in sleep timing, sleep duration was unaffected, whereas problems with sleeping in and early morning awakening (dozing in bed) were more prevalent during winter only. Hence, sleep efficiency was poorest during the dark period. Another finding coinciding with Friborg et al. [2] was that seasonality in sleep was more prevalent for work days than for weekend days. To the authors' knowledge, seasonality in chronotype has not been studied prospectively before. We recorded a delay in chronotype of about 32 min (95% confidence interval, 14–50) from autumn to winter, which was much more pronounced than in the population-based 'Tromsø 6' study (~10 min) [5]. Despite its large sample size ($N = 12{,}984$), the Tromsø 6 study may suffer from more measurement errors (retrospective recall and a single rating) and a methodologically weaker design (cross-sectional).

The novel findings of the present study were that seasonality in sleep was modified by elevated levels of depression. Moreover, we also showed that anxiety and fatigue acted as less reliable modifiers, and that other measures of psychological self-regulation or regularity in daily habits (ie eating and physical activity) did not modify the relationship between seasonality and sleep timing.

4.1. Seasonal changes in insomnia, fatigue, emotional and physical health

Regarding the questionnaire data, we found comparable seasonality in measures of insomnia (mostly daytime problems compared with nocturnal symptoms), fatigue (mostly physical rather than mental fatigue), negative mood (depression to a stronger degree than anxiety), subjective health complaints, physical activity, and school-related stress (attendance and performance to an equal degree). Several of these findings also replicated previous findings [1,2,18]. Due to moderate to strong correlations between the questionnaire measures, additional multivariate regression analyses were performed. After adjusting for the influence of these variables on each other, fatigue was the only variable still showing variability across seasons. This does not imply that the other variables do not indicate seasonality, but that fatigue is the one factor showing the strongest seasonal variation compared with all the others. Moreover, the presence of physical fatigue modified seasonality in sleep more strongly than mental fatigue, also replicating previous findings in the Arctic [2]. An interesting distinction was that depression, which showed less seasonality than fatigue, still modified seasonality in sleep timing more strongly than fatigue. This underscores the potential importance of preventing depression to maintain stable circadian sleep rhythms.

4.2. Possible causal mechanisms

Depressed mood was the most potent modifier of sleep timing. Individuals with no or low levels of depressive symptoms appear to self-regulate their sleep timing quite well during the dark period. This begs an obvious question: what might be the underlying mechanism? One explanation may relate to the suprachiasma nuclei (SCN), which may change its circadian activity in relation to varying degrees of depression [56]. A recent study showed that depressed patients had considerable upregulation of MT1 but not MT2 receptors in the SCN [57]. But as the latter may play a role as a mediator of phase-shifting effects of melatonin [58], future studies of circadian sleep disturbances in depressed patients in the Arctic should consider extracting RNA in addition to sleep log data to examine the role of these receptors in humans. Polymorphisms in several clock genes (eg Period2, Period3, NPAS2, and CLOCK) may also be potential reasons as these too are associated with susceptibility for developing depression [59,60].

Alternatively, it has been theorized that depressed individuals may suffer from abnormalities in circadian rhythmicity due to disruptions caused by stressful life events (the Social Zeitgebers theory [61]). Social rhythms may in turn be affected (e.g. personal relationships, eating, and physical activity), as well as other somatic symptoms [56]. We did not measure all these factors, but it is possible that our measurement of depression included some associated variance with these above-mentioned factors. Including assessments of stressful life events and other elements of individuals' social rhythms would have strengthened our findings. However, as we measured study-related stress this objection may be less strong. A final caveat relates to the lack of data on students' working nights and to whether it covaried with seasons, particularly during examination periods in December. However, as about one-third of the students had no examinations in December, and half of the remaining students had finished their examinations before starting the sleep diary [62], this concern is lessened. Nor were the variance estimates for bed times significantly different across seasons.

The null correlation between depressive symptoms and sleep duration was an unexpected finding in the present study, whereas the 'young HUNT' study in Norway ($N = 9875$) [63], for example, found shorter sleep duration among depressed adolescents. The sleep parameters in the present study contributing to these differences were WASO and SOL. One reason may be that our study included very few subjects with moderate or major depressive problems, hence precluding the use of established cut-off criteria [45] to isolate a group of markedly 'depressed' subjects, as was done in the study by Sivertsen et al. [63].

Nevertheless, we found that low levels of depression in a healthy student sample were associated with seasonality in sleep timing. The seasonality reported here also concurs with treatment studies showing that bright light administered in the morning may alleviate symptoms of SAD [64,65]. In northern Norway, SAD is considered a controversial diagnosis, as general population studies based on five different samples ($N \approx 30{,}000$) have failed to find firm evidence of SAD [66,67]; however, we lack studies on the prevalence and role of SAD among psychiatric treatment-seeking samples. An issue for future research is therefore to explore the impact of higher levels of depression on sleep timing. This may be studied by examining seasonal changes in sleep, sleep timing, and chronotype in clinical samples with a mood disorder diagnosis residing within the Polar region, including extraction of melatonin and mRNA. Diagnostic assessments of major depression in addition to a seasonal mood pattern would provide important information about the role of these respective conditions with regard to phase delays in sleep during the two months of darkness in the city of Tromsø.

4.3. Limitations and future directions

The present study is not without its limitations. Season is obviously not possible to manipulate experimentally, so any causal conclusions are precluded. However, the use of prospective measurements of sleep timing represents a stronger design by reducing recall biases [68], error variance related to individual differences, and carry-over effects if counterbalancing designs are used (which was partly employed in the present study). These findings would still be strengthened by simultaneous measurement with wrist actigraphy, which has been validated for sleep timing (onset and offset [69]). Actigraphy might also collect data about the intensity of ambient indoor evening light across seasons, and provide statistical model adjustments. However, as indoor daylight is much brighter during summer evenings than during winter evenings, the present findings could in theory have been strengthened if such an adjustment had been available.

Similarly, objective measures of circadian phase markers, such as body temperature or dim light melatonin onset, would have confirmed that the endogenous circadian rhythm is influenced by seasonal changes in bright morning daylight. As melatonin is highly affected by light and darkness, changes in dim light melatonin onset would be an excellent indicator of seasonal circadian changes in Polar Arctic populations [15,16]. Collecting additional objective data on circadian rhythmicity would provide a stronger test of whether impaired self-regulation or circadian misalignment is the main mediator of seasonality in sleep timing and chronotype.

5. Conclusions

The study of seasonal effects on sleep in Polar regions, including Tromsø at 69°N, began in the 1950s [70], yet relatively few studies have been conducted. The present findings provide not only prospective confirmation that sleep timing is delayed during the dark period (December) compared to seasons with brighter photoperiods (September and March), but also that seasonal sleep effects are modified by depression, and to a lesser extent by anxiety and fatigue. Contrary to the hypotheses, behavioral regularity in eating habits and physical activity, or psychological self-regulation, did not modify seasonality in sleep timing. Future research is needed to confirm these influences on sleep timing in order to develop effective interventions to improve the sleep health of individuals residing in the Polar regions.

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Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.03.014.

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